Many signaling molecules exhibit complex dynamical behaviors, yet little is known how these dynamics affect cellular responses. In this talk I will focus on the dynamics of p53, a critical tumor-suppressive protein that controls genomic integrity and cell survival. The dynamics of p53 depends on the stimulus. In response to UV p53 shows a single graded pulse while in response to double strand breaks caused by \( \gamma \) irradiation p53 shows a series of repeated pulses. Using a computational model, we identified a sequence of precisely timed drug additions that switch p53 dynamics from pulsatile into sustained, and we tested the resulting behavior in cells. Our results show that different dynamical patterns of p53 alter the selection and timing of gene expression and perturb cellular outcomes in response to DNA damage. More broadly, our results show that protein dynamics can be an important part of a signal, directly influencing cell fate decisions.